

**California Environmental Protection Agency**



**Air Resources Board**

**INITIAL STATEMENT OF REASONS**  
**FOR RULEMAKING**

**STAFF REPORT**

**PROPOSED IDENTIFICATION OF DIESEL EXHAUST**  
**AS A TOXIC AIR CONTAMINANT**

**Prepared by the Staff of**  
**the Air Resources Board and**  
**the Office of Environmental Health Hazard Assessment**

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# **INITIAL STATEMENT OF REASONS FOR RULEMAKING**

## **STAFF REPORT**

### **INTRODUCTION**

In accordance with California Health and Safety Code sections 39660-39662, the Air Resources Board (ARB or Board) staff is recommending that the Board identify diesel exhaust as a toxic air contaminant (TAC).

This Initial Statement of Reasons for Rulemaking for the Proposed Identification of Diesel Exhaust as a TAC consists of:

- 1) an ARB Staff Report, which summarizes the scientific basis for the proposed regulation and includes a discussion of the environmental and economic impacts of the proposal;
- 2) Appendix I (the Proposed Regulation Order);
- 3) Appendix II (the Findings of the Scientific Review Panel [SRP]); and
- 4) Appendix III (the SRP-approved version of the Executive Summary and the three-part report that contains the analysis of the exposure and health assessments of diesel exhaust, Parts A, B, and C, as approved on April 22, 1998).

Part A, prepared by the ARB staff, is an evaluation of emissions of, and exposure to, diesel exhaust. Part B, prepared by the Office of Environmental Health Hazard Assessment (OEHHA) staff, assesses the health effects of diesel exhaust. Part C consists of copies of the public comments received on the June 1994, May 1997, and February 1998 draft reports, and the ARB/OEHHA staff responses.

### **BACKGROUND**

#### **1. Definition of a Toxic Air Contaminant**

Section 39655 of the California Health and Safety Code defines a TAC as “an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.” California Health and Safety Code section 39013 defines air pollutant as “any discharge, release, or other propagation into the atmosphere and includes, but is not limited to, smoke, charred paper, dust, soot, grime, carbon, fumes, gases, odors, particulate matter, acids, or any combination thereof.” In addition, substances which have been listed as federal hazardous air pollutants (HAPs) pursuant to section 7412 of Title 42 of the United States Code have also been identified as TACs under the state's air toxics program pursuant to section 39657(b) of the California Health and Safety Code.

## **2. The California Program for Identification and Control of Toxic Air Contaminants**

This program was established by a California law, Assembly Bill 1807 (AB 1807, Tanner, Chapter 1047, statutes of 1983, Health and Safety Code section 39650 *et seq.*, Food and Agriculture Code section 14021 *et seq.*). AB 1807 created a comprehensive program administered by the ARB to reduce the potential adverse public health impacts caused by emissions of toxic substances to the ambient air.

AB 1807 established a two-phased process which separates risk assessment (identification) from risk management (control). During the identification phase, a report is developed which determines whether there may be potential adverse health effects from substances in consideration of their toxicity, quantities of emissions and human exposure in California. If the Board formally identifies a substance as a TAC, the substance enters the risk management phase. In the risk management phase, the ARB determines the need for and appropriate degree of controls in consideration of cost and potential health benefits. The identification phase and the control phase are open public processes in which the ARB staff actively seeks industry and public participation. (Health and Safety Code section 39660-39666.)

In setting priorities for substances to enter the AB 1807 identification process, the ARB must consider factors relating to: 1) risk of harm to public health; 2) amount or potential amount of emissions; 3) manner of, exposure to, and usage of the substance in California; 4) persistence in the atmosphere; and 5) ambient concentrations in the community. (Health and Safety Code section 39660(f).)

Once a substance is entered into the identification process, the ARB and OEHHA staffs prepare a report that serves as the basis for listing the substance as a TAC. Health and Safety Code section 39660 requires the OEHHA, upon the request of the ARB, to evaluate the health effects of a potential TAC while the ARB evaluates the exposure data associated with it.

The ARB's exposure assessment is based, to the extent available, upon research and monitoring data, emissions inventory data, toxic chemical release data, and information on estimated actual exposures from data on ambient and indoor air environments. (Health and Safety Code section 39660(f).)

The OEHHA's health evaluation includes an assessment of the availability and quality of data on the substance's health effects, including its potency and mode of action. Where it can be established that a threshold of adverse health effects exists, the OEHHA must identify a safe exposure level. If there is no threshold of significant adverse health effects, a range of risk for exposure to the substance is determined. (Health and Safety Code section 39660(c).) In both cases, care is taken by the OEHHA to provide a full explanation of any uncertainties associated with the data.

The report, together with the scientific data on which the report is based, is made available to the public and is formally reviewed by the SRP pursuant to Health and Safety Code section 39661. The SRP is composed of nine members, one each with recognized scientific expertise in the field of oncology, pathology, epidemiology, atmospheric science, biostatistics, occupational medicine, toxicology, and biochemistry (or molecular biology), and one member with relevant scientific experience who is experienced in the operation of scientific review or advisory bodies. The SRP reviews the scientific procedures and methods used to support the data, the data itself, and the conclusions and assessments on which the report is based. The SRP conducts all of its business at noticed meetings which are open to the public.

If the SRP approves the report, it will prepare and adopt formal findings. If the SRP determines that the report is not based on sound scientific knowledge, methods, or practices, it will return the report for revision and resubmittal. Once the SRP has reviewed and approved a report, it transmits the report with the SRP-adopted findings to the ARB. The Board conducts a public hearing to determine, based on the staff's report and the SRP's findings, if a substance should be listed as a TAC. If the Board decides to list the substance as a TAC, it is added to section 93000 of the California Code of Regulations. Health and Safety Code section 39665(a) then requires the ARB to prepare a report which assesses the need for and appropriate degree of control for that substance.

## **DEVELOPMENT OF THE REPORT ON DIESEL EXHAUST**

Diesel exhaust entered the AB 1807 process in October 1989 and has undergone an extensive evaluation. Diesel exhaust was entered into the process because it has potential cancer and noncancer health effects and widespread exposure in California. The International Agency for Research on Cancer (IARC) had listed diesel exhaust as a "probable" human carcinogen and the U.S. Environmental Protection Agency (U.S. EPA) had begun an evaluation of both the cancer and noncancer health effects. The ARB and the OEHHA gave priority to the evaluation of diesel exhaust because it met the TAC program criteria related to potential risk of harm to public health, amount of emissions, exposure and use, and persistence in the atmosphere.

In March 1990, the ARB sponsored a conference on the risk assessment of diesel exhaust. On June 17, 1994, the first draft TAC identification report was released to the public for a six-month comment period. On September 14, 1994, a public workshop was held to discuss the report. On January 29-30, 1996, the OEHHA, the ARB, the Health Effects Institute (HEI), the National Institute for Occupational Safety and Health, the World Health Organization, and the U.S. EPA sponsored a scientific workshop to discuss the application of human health study data in developing quantitative cancer risk estimates for diesel exhaust.

A second draft report, which was revised to incorporate public comments received during the first public comment period (June 1994-December 1994), was released for public comment in May 1997. On July 1, 1997, a third public workshop was held to discuss the revised draft report. SRP members participated in these workshops. On October 16, 1997, the SRP held a meeting to

discuss the comments received on this draft report. A third draft report was released for public comment on February 23, 1998. The SRP held a meeting on March 11, 1998, to hear from invited scientists who have expertise in the study of diesel exhaust. On April 22, 1998, the SRP reviewed and approved the draft report and adopted its findings. Appendix II contains the SRP's findings as adopted at the April 22, 1998, meeting. Throughout this process, the ARB and OEHHA staffs conducted numerous other meetings and conference calls with the affected industries, associations, environmental groups, governmental agencies, and other interested parties.

## **DIESEL EXHAUST EMISSIONS AND EXPOSURE IN CALIFORNIA**

The ARB staff reviewed and evaluated the potential for diesel exhaust exposure in California. The ARB staff considered the estimated emissions inventory and ambient concentrations. The SRP approved the ARB staff's exposure assessment at its April 22, 1998, meeting.

### **1. Emissions**

Diesel exhaust is a complex mixture of inorganic and organic compounds that exist in gaseous, liquid, and solid phases. As with combustion of other fuels, the primary gaseous components are nitrogen, oxygen, carbon dioxide, and water vapor. Diesel exhaust includes over 40 substances that are listed as hazardous air pollutants by the U.S. EPA and by the ARB as TACs. The substances listed below have either been detected or predicted to occur in the exhausts of diesel engines based on observed chemical reactions and/or their presence in the fuel or lubricating oil. Fifteen of these substances are listed by the IARC as carcinogenic to humans, or as a probable or possible human carcinogen. Further research is needed to determine the contribution of many of these substances to atmospheric diesel exhaust exposures.

The combustion of diesel fuel in a compression ignition engine produces diesel exhaust. Approximately 2.1 billion gallons of diesel fuel were burned in such engines in California during 1995. In the future, estimated diesel fuel consumption is predicted to increase in California from current levels up to 2.3 billion gallons in 2000 and to 2.9 billion gallons in 2010.

One of the main characteristics of diesel exhaust emitted from newer diesel engines is the release of particles at a rate of about 20 times greater than from comparable gasoline engines. Diesel exhaust particles carry many of the organics and metals present in the exhaust. Over 90 percent of the mass of these particles are less than 2.5 microns in diameter. Because of their small size, these particles are easily inhaled into the bronchial and alveolar regions of the lung.

There are three groups of sources of diesel exhaust in California: mobile sources (on-road vehicles and other mobile sources), stationary area sources (e.g., oil and gas production facilities, stationary engines, shipyards, repair yards), and stationary point sources (e.g., chemical manufacturing, electric utilities). In 1995, emissions of oxides of nitrogen (NO<sub>x</sub>) from diesel fuel use were about 415,000 tons per year (tpy); oxides of sulfur (SO<sub>x</sub>) about 28,000 tpy; and reactive

## Substances in Diesel Exhaust Listed by the ARB as Toxic Air Contaminants

acetaldehyde	hexane
acrolein	inorganic lead
aniline	manganese compounds
antimony compounds	mercury compounds
arsenic	methanol
benzene	methyl ethyl ketone
beryllium compounds	naphthalene
biphenyl	nickel
bis[2-ethylhexyl]phthalate	4-nitrobiphenyl
1,3-butadiene	phenol
cadmium	phosphorus
chlorine	POM, including PAHs and their derivatives
chlorobenzene	propionaldehyde
chromium compounds	selenium compounds
cobalt compounds	styrene
cresol isomers	toluene
cyanide compounds	xylene isomers and mixtures
dioxins and dibenzofurans	o-xylenes
dibutylphthalate	m-xylenes
ethyl benzene	p-xylenes
formaldehyde	

organic gases (ROG) about 41,000 tpy. Diesel exhaust  $PM_{10}$  (particulate matter equal to or less than 10 microns in diameter) and  $PM_{2.5}$  (particulate matter equal to or less than 2.5 microns in diameter) emissions during 1995 were estimated to be about 27,000 tpy and 26,000 tpy, respectively. Statewide emission estimates of other substances emitted from diesel exhaust (such as benzene, formaldehyde, and 1,3-butadiene) have not been quantified. Further research is needed to accurately estimate diesel exhaust-associated emissions of these substances.

Over the past 20 years, advances in engine design and fuel formulation have been made as a result of control measures enacted by the ARB and U.S. EPA and, in the case of engine design, to improve efficiency. As a measure of the effectiveness of these standards and regulations, statewide diesel exhaust  $PM_{10}$  emissions from on-road mobile sources are expected to be reduced by approximately 60 percent between 1995 and 2010. However, unless further controls are established, emissions are expected to increase by about 10 percent from 2010 through 2020 due to increases in the number of vehicles and usage. Because less stringent standards have been applied to off-road engines, total diesel exhaust emissions from on-road and off-road mobile sources are expected to decrease by about 30 percent between 1995 and 2010.

## 2. Exposure

Our assessment of population exposure to diesel exhaust focuses on the PM<sub>10</sub> component because more is known about the particulate matter fraction of diesel exhaust. Diesel exhaust particles contain many of the toxic components of the exhaust. We have a complete emissions inventory and ambient concentration data base for diesel exhaust-derived particulate matter in California. In addition, particulate matter has been associated with approximately 50 to 90 percent of the mutagenic potency of whole diesel exhaust.

However, in our review of the health effects of exposure to diesel exhaust, we are examining the overall toxicity of the exhaust. This is because the exposures experienced in the occupational human health studies have been to the whole exhaust. The IARC, the National Institute for Occupational Safety and Health, and the U.S. EPA have also evaluated diesel exhaust this way. We believe this approach is necessary because it is most consistent with the scientific studies to assess health effects from exposure to diesel exhaust. It is recognized that the compounds of concern are those that are emitted as a result of the incomplete combustion of diesel fuel.

The staff estimated a population-weighted average outdoor diesel exhaust PM<sub>10</sub> exposure concentration in California in 1995 to be 2.2 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ) (see Appendix III, Part A). Several independent studies have reported similar outdoor air diesel exhaust PM<sub>10</sub> concentrations. The 1995 average indoor exposure concentration was estimated to be about 1.47  $\mu\text{g}/\text{m}^3$ , which takes into account building air exchange rates, adult and children's activity patterns, and air concentrations of diesel exhaust particles. The 1995 population time-weighted average total air exposure to diesel exhaust PM<sub>10</sub> concentrations across all environments (including outdoors) is estimated to be 1.54  $\mu\text{g}/\text{m}^3$  or, when rounded, 1.5  $\mu\text{g}/\text{m}^3$ . Emission reductions that had occurred by 1995 from adopted new engine emission standards and diesel fuel reformulation were taken into account in calculating these exposure numbers.

Higher level, near-source exposures to diesel exhaust occur near busy roads and intersections where many diesel vehicles are operating. Diesel exhaust PM<sub>10</sub> concentrations as high as 10  $\mu\text{g}/\text{m}^3$  over a 24-hour period have been measured near a freeway. This is about five times the 1995 outdoor ambient air concentration of 2.2  $\mu\text{g}/\text{m}^3$  and about six times the 1995 total air exposure estimate of 1.5  $\mu\text{g}/\text{m}^3$ .

As discussed previously, diesel exhaust is a complex mixture of substances, and each substance will remain in the air or react with other substances according to the substance's individual chemical properties. Diesel particles are typically smaller than 1 micron and are expected to remain in the air for about 10 days, provided that the weather is dry. Diesel particles are effectively removed by precipitation and persist for much shorter periods during these events.

Diesel exhaust emissions can deposit onto water, soil, and vegetation. Exposure by ingestion and dermal absorption can occur through these other media and through airborne exposures to re-entrained dust. In order to assess non-inhalation pathways, substance and site-specific data are

needed. Since the specific parameters for diesel exhaust are not known for these pathways, a multi-pathway assessment for diesel exhaust exposure was not done. Therefore, our estimates of exposure to diesel exhaust are conservative and underestimate the public's actual exposure to diesel exhaust. More research is needed in this area for diesel exhaust.

As a result of the emission reductions described previously, ambient levels of diesel exhaust  $PM_{10}$  are expected to be reduced by about 20 percent from 1995 to 2010.

### **3. Diesel Exhaust Constituents from Pre-1993 Fuel and Post-1993 Fuel Combustion**

To address the effects that recent changes in diesel fuel composition may have on the toxic exhaust constituents from newer diesel engines, the ARB funded a study by the College of Engineering, Center for Environmental Research and Technology (CE-CERT) at the University of California, Riverside. The study design included the testing of one heavy-duty diesel engine (that was typical of engines on the market in California) using three fuels (a single fuel that meets specifications of low sulfur fuel required before 1993, a very low aromatic fuel, and a fuel designed to be typical of the fuels in use now). The study design did not produce data that could be used to obtain statistically robust conclusions (due to the use of only one engine and the limited number of data points for each target analyte/fuel combination). Testing was conducted from December 1996 to January 1997 and results became available in spring of 1998.

The results from this study show reductions of criteria pollutants consistent with what was estimated by the ARB staff for the 1988 diesel fuel regulation. The low aromatic and typical in-use fuels produced particulate matter and  $NO_x$  exhaust emissions reductions that met the regulation's requirements.

A comparison of the milligram to milligram per brake horsepower-hour emission profiles using the three different fuels showed the presence of the same toxic substances, and a similar distribution of toxic substances in general. The low aromatic and alternative fuels resulted in lower emission rates for some particle- and vapor-phase PAHs including anthracene, benz[a]anthracene, dibenzo[a,h]pyrene, naphthalene, and biphenyl. In addition, higher mutagenic activity was observed in both the particle and vapor phase collected from pre-October 1993 fuel than from the low aromatic and alternative formulation fuels. However, because of the limited testing, it is not possible to determine if the mutagenicity differences are statistically significant.

The CE-CERT study also tested for dioxins with the purpose of identifying and attempting to quantify dioxins for method development. The results show that dioxins are present in exhaust from both the old and the new fuel. However, the results are qualitative only and indicate the need for further method development.

Overall, the results from this study indicate that the diesel exhaust from the new diesel fuel tested contained the same toxic air contaminants as the old fuel, although their concentrations may differ for some compounds. Further research would be necessary to quantify the amounts of

specific compounds emitted from a variety of engine technologies, operating cycles, and fuel to characterize better any differences between old and new fuels and technologies and the potential impact on the toxicity of diesel exhaust.

## **HEALTH EFFECTS OF DIESEL EXHAUST EXPOSURE**

The OEHHA reviewed and evaluated the potential for diesel exhaust to affect human health, and the associated scientific uncertainties. The OEHHA considered acute and chronic noncancer health impacts, and potential cancer health impacts. The SRP approved the OEHHA's health assessment at its April 22, 1998, meeting.

A number of adverse short-term (acute) health effects have been associated with exposures to diesel exhaust. Occupational exposures to diesel exhaust particles have been associated with significant cross-shift decreases in lung function. Increased cough, labored breathing, chest tightness, and wheezing have been associated with exposure to diesel exhaust in bus garage workers. A significant increase in airway resistance and increases in eye and nasal irritation were observed in human volunteers following one-hour chamber exposure to diesel exhaust. In acute and subchronic animal studies, exposure to diesel exhaust particles induced inflammatory airway changes, lung function changes, and increased the animals' susceptibility to infection.

A number of adverse long-term (chronic) noncancer effects have been associated with exposures to diesel exhaust. Occupational studies have shown that there may be a greater incidence of cough, phlegm and chronic bronchitis among those exposed to diesel exhaust than among those not exposed. Histopathological changes in the lung of diesel-exposed test animals reflect inflammation of the lung tissue. Reduced pulmonary function was noted in monkeys during long-term exposure. Reductions in pulmonary function have also been reported following occupational exposures in chronic studies.

Diesel exhaust particles can induce immunological allergic reactions and localized inflammatory responses in humans, as well as acting as an adjuvant for pollen allergy. Intranasal challenge with diesel exhaust particles in human volunteers resulted in an immunological response. Co-exposure to diesel exhaust particles and ragweed pollen resulted in an immune response greater than that following pollen or diesel exhaust particles alone. Effects of intratracheal, intranasal, and inhalation exposures of laboratory animals are supportive of the findings in humans. These effects include allergic reactions and inflammation, increased mucus secretion and respiratory resistance, and airway constriction.

The World Health Organization and the OEHHA have conducted further analyses of the dose-response relationships for several of the non-cancer, adverse effects of chronic exposures to diesel exhaust on the rat lung. These analyses gave a range of health risk guidance values of 2 to 21  $\mu\text{g}/\text{m}^3$  and support the adoption of 5  $\mu\text{g}/\text{m}^3$  which is also the 1993 U.S. EPA Reference Concentration. A U.S. EPA Reference Concentration or California Reference Exposure Level (REL) of a chemical is an estimate, with uncertainty spanning perhaps an order of magnitude, of the air concentration below which no noncancer adverse health effects are likely to occur from

lifetime exposure. This estimate takes into consideration persons who may be more sensitive than others to the effects of a chemical. The OEHHA concurs with the U.S. EPA in recommending 5  $\mu\text{g}/\text{m}^3$  as the chronic REL for diesel exhaust.

Diesel exhaust contains genotoxic compounds in both the vapor phase and the particle phase. Diesel exhaust particles or extracts of diesel exhaust particles are mutagenic in bacteria and in mammalian cell systems, and can induce adverse chromosomal changes. DNA adducts (representing genotoxins bound chemically to DNA) have been shown to increase following inhalation exposure of rodents and monkeys to whole diesel exhaust and have been found in mammalian cells following treatment with diesel exhaust particle extract. Elevated levels of DNA adducts have been associated with occupational exposure to diesel exhaust.

Over 30 human epidemiological studies have investigated the potential carcinogenicity of diesel exhaust. These epidemiological studies provide evidence consistent with a causal relationship between occupational diesel exhaust exposure and lung cancer. These studies, on average, found that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer. The OEHHA analyzed the lung cancer findings for consistency and found that the association was unlikely to be due to bias or chance. Results of inhalation bioassays in the rat, and with less certainty in mice, have demonstrated the carcinogenic potential of diesel exhaust in animals, although the mechanisms by which diesel exhaust induces lung tumors in animals remain uncertain.

Other agencies or scientific bodies have studied the health effects of diesel exhaust. The National Institute of Occupational Safety and Health first recommended that whole diesel exhaust be regarded as a potential occupational carcinogen based upon animal and human evidence in 1988. The IARC concluded that diesel engine exhaust is probably carcinogenic to humans (Group 2A). Based upon the IARC findings, in 1990, the State of California under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) identified diesel exhaust as a chemical “known to the State to cause cancer.” (Title 22, California Code of Regulations, section 12000.) The 1998 draft U.S. EPA document (Health Assessment Document for Diesel Emissions, Review Draft, February 1998) similarly concluded that diesel exhaust be considered a “probable” human carcinogen (category B1). This conclusion evolves from positive yet “limited” evidence in the human studies, a “sufficient” level of evidence in bioassays, and consideration of the supporting information from mutagenicity and genotoxicity data.

Risk assessments can use carcinogenicity data from either animal or human studies. For diesel exhaust, there are data from human epidemiological studies of occupationally exposed populations which are useful for quantitative risk assessment. On balance, the OEHHA concluded that available human data lend more confidence in the prediction of human risks than the data from the available animal studies because of the uncertainties in the animal studies and of extrapolating from animals to humans. Thus, the OEHHA preferred to derive the range of human risk estimates based only upon the epidemiological findings and not the animal data. Using data from a case-control study and a cohort study, the OEHHA estimated the risk (95 percent upper confidence limit) of lung cancer in the general population due to diesel exhaust. Because of

uncertainties in the actual workplace exposures, the OEHHA developed a variety of exposure scenarios to bracket the exposures that were plausible. Based on these exposure estimates, presented in Table 1-1, the range of resulting estimates of cancer unit risk is  $1.3 \times 10^{-4}$  to  $2.4 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$ . The unit risk represents the 95 percent upper confidence limit of cancer risk per million people exposed per microgram of diesel exhaust particulate in a cubic meter of air over a 70-year lifetime. The SRP approved the range of risk estimated by the OEHHA. In addition, the SRP concluded that a value of  $3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$  is a reasonable estimate of unit risk expressed in terms of diesel particulate (see Appendix II).

The OEHHA and ARB staffs recognize that the limited exposure information available contributes to the uncertainty of the dose response risk assessment based on the human studies. However, the overall magnitude of uncertainty is not atypical of the types of uncertainty encountered when the Board identified other TACs. The greater than usual uncertainty in the exposure estimates is substantially offset by the much smaller than usual range of extrapolation from the occupational exposures to the ambient air concentrations. Interspecies extrapolation uncertainty is not an issue in this diesel exhaust risk assessment. In addition, there are more than 30 human studies of more than one occupation that show overall an increase in lung cancer from diesel exhaust exposure.

Based on available scientific evidence, a level of diesel exhaust exposure below which no carcinogenic effects are anticipated has not been identified. This finding was approved by the SRP at its meeting on April 22, 1998.

As with other substances evaluated by the SRP and after reviewing the field of published peer reviewed research studies on diesel exhaust, the SRP indicated that additional research is appropriate to further clarify the health effects of diesel exhaust. The OEHHA and ARB staffs recognize that diesel exhaust health studies will continue. For example, the HEI, which is jointly funded by industry and the U.S. EPA, has started a five-year study to review key epidemiologic studies and make recommendations for the design of new studies. The OEHHA and ARB staffs will follow these efforts closely, and will provide support to the extent resources are available. If the outcome of this, or other future health studies, ultimately reduces uncertainties or improves the scientific basis for estimating diesel exhaust risk, the OEHHA and ARB staffs would consider such information. When research results become available, the TAC program has a process in place for further evaluation of new scientific evidence pertaining to a previously completed TAC risk assessment. The process specifically addresses the evaluation and response to submittals of new scientific information as evidence for review of a TAC risk assessment.

**Table 1-1. Summary of Cancer Unit Risks According to Study, Exposure Assumptions, and Modeling Approaches.**

	95% UCL Cancer Unit Risk ( $\mu\text{g}/\text{m}^3$ ) <sup>-1</sup>	95% Upper Confidence Limit of Cancer Risk per Million per Microgram of Diesel Exhaust Particulate in a Cubic Meter of Air Exposure Over a 70-year Lifetime
Garshick <i>et al.</i> (1987a) Case Control <sup>1</sup>		
Scenario <sup>2</sup>		
A	$2.4 \times 10^{-3}$	2400
B	$1.8 \times 10^{-3}$	1800
C	$1.0 \times 10^{-3}$	1000
D	$6.6 \times 10^{-4}$	660
E	$3.6 \times 10^{-4}$	360
Garshick <i>et al.</i> (1988) Cohort Study (Chapter 7) <sup>3</sup>		
Scenario		
A	$1.8 \times 10^{-3}$	1800
B	$1.4 \times 10^{-3}$	1400
C	$8.2 \times 10^{-4}$	820
D	$5.1 \times 10^{-4}$	510
E	$2.8 \times 10^{-4}$	280
Garshick <i>et al.</i> (1988) Cohort Study (Appendix D) <sup>4</sup>		
Scenario A		
general multiplicative model	$1.9 \times 10^{-3}$	1900
biologically based <sup>5</sup>	$3.8 \times 10^{-4}$	380
Scenario C		
general multiplicative model	$7.2 \times 10^{-4}$	720
biologically based <sup>5</sup>	$1.3 \times 10^{-4}$	130
biologically based <sup>6</sup>	$1.5 \times 10^{-4}$	150

**EVALUATION OF NEED AND APPROPRIATE DEGREE OF CONTROL FOR DIESEL**

<sup>1</sup> Using published slope coefficient for hazard on years to diesel exhaust as described in Appendix III (Part B, Section 7.3.3).

<sup>2</sup> A Ramp pattern of exposure plateauing in 1959 at the 1980 exposure level of  $50 \mu\text{g}/\text{m}^3$   
 B Roof pattern of exposure peaking in 1959 at twice the 1980 exposure level of  $40 \mu\text{g}/\text{m}^3$   
 C Roof pattern of exposure peaking in 1959 at 3-fold the 1980 exposure level of  $50 \mu\text{g}/\text{m}^3$   
 D Roof pattern of exposure peaking in 1959 at 3-fold the 1980 exposure level of  $80 \mu\text{g}/\text{m}^3$   
 E Roof pattern of exposure peaking in 1959 at 10-fold the 1980 exposure level of  $50 \mu\text{g}/\text{m}^3$

<sup>3</sup> Using individual data to obtain a slope for hazard on years of exposure to diesel exhaust as described in Appendix III (Part B, Section 7.3.4).

<sup>4</sup> Applying time varying concentrations to individual data to obtain a slope of hazard on exposure as described in Appendix III (Part B, Appendix D).

<sup>5</sup> 6th/7 stage model.

<sup>6</sup> 7th/7 stage model.

## **EXHAUST**

If the Board votes to formally identify diesel exhaust as a TAC, the staff will begin an analysis to determine what if any further regulatory action to reduce exposures to diesel exhaust is necessary. A report will be prepared, in consultation with the local districts, affected industries, and the public, on the need and appropriate degree of control (the “needs assessment”) for diesel exhaust. The report is required by Health and Safety Code section 39665(a) and must include the following information: present and potential future emissions and associated potential risks; atmospheric persistence; number and categories of emission sources; available control technologies; costs for reducing emissions; alternative sources of emission reductions; and the potential adverse health, safety, or environmental impacts associated with the implementation of a control measure.

In its assessment, staff will focus on technological opportunities, beyond those already in place, to reduce further public exposures to emissions from diesel engines. Staff’s focus will be on reducing emissions of those components of diesel exhaust that are emitted as a result of the incomplete combustion of diesel fuel. We anticipate that our focus will be on the particulate matter component, and potentially the volatile organic component, of diesel exhaust. We believe this approach to be consistent with the identification of whole diesel exhaust because we know there are toxic constituents on the particle and in the vapor phase. Staff will not be considering a ban on the use of diesel fuel or diesel engines, because such a strategy would not be technically and economically feasible.

If this needs assessment identifies additional technically feasible and cost-effective opportunities for reducing exposures to diesel emissions within the authority of the ARB or air pollution control districts, the ARB would then design specific control measures in an open regulatory process with full public involvement.

To ensure full opportunity for public consultation and participation in the needs assessment process, the ARB staff intends to create a diesel exhaust risk management working group (working group). Invitations to participate would be sent to interested industries, associations, environmental groups, other governmental agencies such as the U.S. EPA and air districts, and other interested parties.

The working group would advise the ARB staff and serve as a forum for on-going communication, cooperation, and coordination in the identification of additional opportunities to reduce further emissions from diesel engines. Meetings of the group would be publicly noticed and the meetings would be open to all. We intend to hold an initial organizational meeting of the working group this fall.

Over the past 20 years, a number of regulations have been adopted by the ARB and the

U.S. EPA that have resulted in or will result in significant reductions in emissions of pollutants such as NO<sub>x</sub>, SO<sub>x</sub>, and particulate matter from diesel engines. These include:

- ▶ a requirement for low sulfur/low aromatic diesel fuel that reduces particulate matter, NO<sub>x</sub>, and SO<sub>x</sub> emissions (effective 1993);
- ▶ emission standards that restrict the amount of particulate matter emitted by new diesel cars, trucks, urban buses, and heavy-duty trucks (phased-in from 1982 through 1996);
- ▶ emission standards for NO<sub>x</sub> emissions from diesel vehicles, heavy-duty trucks, and urban buses (phased in from 1984 through 2004);
- ▶ the roadside testing of heavy-duty diesel vehicles for excessive particulate matter emissions (effective 1991) and a requirement for fleet inspection and maintenance of heavy-duty diesel vehicles (anticipated to begin in summer 1998);
- ▶ emission standards that restrict the amount of particulate matter and NO<sub>x</sub> that can be emitted from many 1995 and newer small off-road diesel engines (<25 hp) (effective 1995);
- ▶ emission standards for particulate matter and NO<sub>x</sub> from heavy-duty off-road diesel equipment (>175 hp) (effective 1996); and
- ▶ a requirement that portable diesel engines registered in the Statewide Portable Equipment Program must satisfy, by 2010, the above emission standards for heavy-duty off-road diesel equipment (>175 hp) and federal standards for particulate matter and NO<sub>x</sub> from heavy-duty off-road diesel equipment (>50 hp) (effective 1997).

Significant progress has been made as a result of the above emissions standards and fuel regulations. For example, since 1988, particulate matter emissions from new heavy-duty diesel truck engines have been reduced by over 85 percent, and from urban bus engines by over 90 percent. Emissions of NO<sub>x</sub> from heavy-duty diesel truck engines have also been reduced since the mid-1980s by over 20 percent. SO<sub>x</sub> emissions have also decreased due to the reduction of sulfur content in fuels by over 80 percent in 1993.

In addition, the ARB staff is working on the following measures to reduce emissions from diesel engines:

- ▶ fuel specifications for locomotives;
- ▶ more stringent standards for light- and medium-duty diesel vehicles;
- ▶ more stringent standards (in conjunction with U.S. EPA) for all off-road diesel equipment;
- ▶ more stringent standards (in conjunction with U.S. EPA) for locomotives operating in the

South Coast Air Basin in 2010 and beyond;

- ▶ incentive measures for new and in-use on-road diesel vehicles and off-road diesel engines that could provide a boost for alternative fuel engine introduction.

Part of the effort of the working group would be to assess whether additional control measures are needed in light of the impact of existing and planned regulations on diesel exhaust emission trends and exposures. Technical work already planned in connection with new federal fine particulate matter standards will help quantify the sources of diesel particulate matter and the effect of the ARB's current control program. If a need for further control is found, the ARB staff would examine strategies such as tighter emission standards for new vehicles; further reformulation of diesel fuel; maintaining low emissions in-use; and incentive programs to promote accelerated turnover of in-use diesel engines and alternative engine and fuel technologies in urban areas where the vast majority of population exposure to diesel exhaust occurs. An incentive program, such as that proposed by the Governor for the 1998-1999 budget, could provide a boost for alternative fuel engine introduction and engine replacement with cleaner engines.

## **ENVIRONMENTAL IMPACT ASSESSMENT**

The identification of diesel exhaust as a TAC is not expected to result in any adverse impact on the environment. The Board's identification of diesel exhaust as a TAC and the subsequent analysis of the need to control emissions in the AB 1807 risk management program may result in the adoption of control measures pursuant to Health and Safety Code sections 39665 and 39666. When considering the adoption of control measures, the ARB will consider all potential impacts of the measures on human health, as well as the potential benefits to public health by reducing diesel exhaust emissions. Therefore, the identification of diesel exhaust as a TAC may ultimately result in control measures that will result in environmental benefits. Adverse environmental impacts identified with respect to specific control measures will be included in the consideration of such control measures pursuant to Health and Safety Code sections 39665 and 39666.

## **ECONOMIC IMPACT ASSESSMENT**

The identification of diesel exhaust as a TAC will not directly have any economic impact on mobile sources of diesel exhaust because the act of identifying a TAC does not mandate any specific risk management action. Once a substance is identified, the ARB is required to assess the need and appropriate degree of control for that substance. Potential control measures for both mobile and stationary sources will be assessed and developed in a full public forum in which the impact of these measures on businesses would be fully assessed.

While most of the diesel exhaust emitted to California's atmosphere is a result of the incomplete combustion of diesel fuel in mobile sources, diesel exhaust emissions also occur from

stationary facilities using diesel fuel. Identifying diesel exhaust as a TAC could have an economic impact on these types of stationary sources as they comply with district rules and permit requirements. Districts have the authority to require that public exposure to particular toxic substances not exceed levels deemed by the district to be protective of public health. Some districts' permitting requirements specify that no new or modified facilities exceed a specific risk level based on the SRP-approved cancer risk numbers for specific toxic substances. These requirements may result in facility operators needing to purchase control equipment or being denied a permit.

Districts are required to implement the AB 2588 Air Toxics Hot Spots Information and Assessment Act of 1987 (AB 2588; Health and Safety Code sections 44300-44384.) This Act establishes a program to inventory emissions of toxic air pollutants and to assess the risk to public health caused by these emissions. Under this program, districts prioritize, require risk assessments, and determine which facilities must notify the public of potential health risks posed by emissions of toxic substances that are on the Hot Spots toxic substances list. If a facility's emissions present a significant health risk, it must develop an Audit and Plan for reducing the risk. The program is supported by fees paid by the affected industries. The fees are specified by state and district regulations which are updated each year through a public process, including public workshops and public hearings.

Diesel exhaust is already on the Hot Spots toxic substances list, and qualified facilities have been required to report their emissions of diesel exhaust and pay program fees. However, the health impacts of diesel exhaust have not been evaluated or quantified under the Program up to this point due to lack of a cancer potency value. The diesel exhaust TAC identification report includes a range of cancer potency values that districts could use to evaluate the cancer risk from sources emitting diesel exhaust. Some facilities may have to prepare risk assessments and, depending on the results, may be required by the districts to notify nearby residents of their emissions. Facilities with large numbers of engines, or with very large engines, and which are located close to populated areas might be affected by these requirements. For example, a stationary diesel engine of approximately 300 horsepower, operating 24 hours per day, 365 days per year, with an emission release point 9 feet above the ground, may exceed a risk of 10 per million. This is only a rough estimate, as the estimated risk will vary with site-specific meteorological conditions, hours of operation, stack height, and control equipment.

There may be Hot Spots fee increases if a facility's potential cancer health risk is equal to or greater than 10 per million. Facilities having a potential cancer risk equal to or greater than 100 per million may need to purchase control equipment or change their operations to reduce exposures to nearby residents.

Some districts include substances listed in the AB 2588 Air Toxics Hot Spots program in their list of sources and substances affected by permitting rules. Other districts may add diesel exhaust upon its identification as a TAC. If districts add facilities that emit diesel exhaust to their permit programs, the affected facilities may be required to pay additional permitting fees. Districts must

conduct socioeconomic analyses, as well as public workshops and hearings, before identified TACs are added to their programs.

Health and Safety Code section 42311 authorizes local air pollution control districts to assess permit fees to recover the cost of district programs required or authorized by state law. Most districts charge facilities fees to cover the cost of developing and implementing the controls required by Health and Safety Code section 39666. The amount of these fees is generally determined by the district resources needed to implement the control requirements. The fees are established by district rulemaking action, and districts must conduct socioeconomic impact analyses, workshops and public hearings before adopting or revising any fee rule.

### **STAFF RECOMMENDATION AND PLAIN ENGLISH SUMMARY OF THE PROPOSED REGULATION**

Based on the information available on diesel exhaust-induced noncancer and cancer health effects and the results of the risk assessment, and the findings of the SRP, we conclude that diesel exhaust meets the definition of a TAC which is an air pollutant “which may cause or contribute to an increase in mortality and serious illness, or which may pose a present or potential hazard to human health” (Health and Safety Code section 39655). Therefore, staff recommends that the Board adopt the proposed regulation shown in Appendix I identifying diesel exhaust as a TAC with no identified level of exposure below which no carcinogenic effects are anticipated. Staff further recommends that the Board direct staff to begin the risk management phase of the program for diesel exhaust and form a diesel exhaust risk management working group to coordinate efforts with the U.S. EPA, industry, environmental groups, and other interested parties.

**APPENDIX I**

**PROPOSED REGULATION ORDER**

## PROPOSED REGULATION ORDER

Amend Titles 17 and 26, California Code of Regulations, Section 93000 to read as follows:

### **93000. Substances Identified as Toxic Air Contaminants.**

Each substance identified in this section has been determined by the State Board to be a toxic air contaminant as defined in Health and Safety Code Section 39655. If the State Board has found there to be a threshold exposure level below which no significant adverse health effects are anticipated from exposure to the identified substance, that level is specified as the threshold determination. If the Board has found there to be no threshold exposure level below which no significant adverse health effects are anticipated from exposure to the identified substance, a determination of “no threshold” is specified. If the Board has found that there is not sufficient available scientific evidence to support the identification of a threshold exposure level, the “Threshold” column specifies “None identified.”

<b><u>Substance</u></b>	<b><u>Threshold Determination</u></b>
Benzene (C <sub>6</sub> H <sub>6</sub> )	None identified
Ethylene Dibromide (BrCH <sub>2</sub> CH <sub>2</sub> Br; 1,2-dibromoethane)	None identified
Ethylene Dichloride (ClCH <sub>2</sub> CH <sub>2</sub> Cl; 1,2-dichloroethane)	None identified
Hexavalent Chromium [Cr(VI)]	None identified
Asbestos [asbestiform varieties of serpentine (chrysotile) riebeckite (crocidolite) cummingtonite-grunerite (amosite), tremolite, actinolite, and anthophyllite]	None identified
Dibenzo-p-dioxins and Dibenzofurans chlorinated in the 2,3,7 and 8 positions and containing 4,5,6, or 7 chlorine atoms	None identified
Cadmium (metallic cadmium and cadmium compounds)	None identified
Carbon Tetrachloride (CCl <sub>4</sub> ; tetrachloromethane)	None identified
Ethylene Oxide (1,2-epoxyethane)	None identified
Methylene Chloride (CH <sub>2</sub> Cl <sub>2</sub> ; Dichloromethane)	None identified
Trichloroethylene	None identified

Chloroform (CHCl <sub>3</sub> )	None identified
Vinyl Chloride (C <sub>2</sub> H <sub>3</sub> Cl; Chloroethylene)	None identified
Inorganic Arsenic	None identified
Nickel	None identified
Perchloroethylene (C <sub>2</sub> Cl <sub>4</sub> ; Tetrachloroethylene)	None identified
Formaldehyde (HCHO)	None identified
1,3-Butadiene (C <sub>4</sub> H <sub>6</sub> )	None identified
Inorganic Lead	None identified
<u>Diesel Exhaust</u>	<u>None identified</u>

NOTE: Authority cited: Sections 39600, 39601, and 39662, Health and Safety Code. Reference: Sections 39650, 39660, 39661, and 39662, Health and Safety Code.

**APPENDIX II**

**FINDINGS OF THE SCIENTIFIC REVIEW PANEL**

May 27, 1998

Mr. John D. Dunlap, III  
Chairman  
Air Resources Board  
2020 L Street  
Sacramento, California 95814

Dear Chairman Dunlap:

I am pleased to forward to you the Scientific Review Panel's (SRP/Panel) Findings (enclosure) for the Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant Report as adopted unanimously at the Panel's April 22, 1998 meeting.

The data, developed and reviewed by OEHHA and ARB, in the scientific risk assessment on exposure to diesel exhaust (Part A) and its health effects (Part B), are extensive and scientifically sound. The SRP notes the report documents the fact that diesel exhaust includes over 40 substances listed by the U.S. Environmental Protection Agency as hazardous air pollutants and by the ARB as toxic air contaminants.

The exposure estimate in the report may underestimate many Californians' actual total exposure because it excludes elevated exposures near roadways, railroad tracks, and inside vehicles. Other routes of exposure to diesel exhaust, such as ingestion and dermal absorption are also excluded.

Development of this report began in 1989, and this compound has the most human epidemiological studies (over 30) than any of the previous 21 toxic air contaminant reports the Panel has reviewed. These studies have investigated the relationship between occupational diesel exhaust exposure and lung cancer, and the epidemiological evidence indicates exposure to diesel exhaust increases the risk of lung cancer. It is noted that in 1990 the State of California, pursuant to Proposition 65, identified diesel exhaust as a chemical "known to the State to cause cancer."

There are a number of adverse long-term noncancer effects associated with exposure to diesel exhaust. These effects include chronic bronchitis, inflammation of lung tissue, thickening of the alveolar walls, immunological allergic reactions, and airway constriction. As new quantitative data emerge from research on adverse noncancer effects from diesel exhaust, the Reference Exposure Level may require adjustment.

John D. Dunlap, III, Chairman  
May 27, 1998  
Page Two

The Panel believes there is still more to be learned about the adverse health effects associated with exposure to diesel exhaust. The Panel is concerned that some technological advances may result in greater total particulate exposure, particularly of fine particles that penetrate deeper into the lungs, but some controls and fuels may reduce overall particulate level. The Panel encourages further research to quantify the amounts of specific compounds emitted from a variety of engine technologies, operating cycles, and fuel to characterize better any differences between old and new fuels and technologies.

The Panel recognizes that diesel exhaust is a mixture of compounds and the potency factor may change as a result of new engine technologies and “cleaner” fuel. Accordingly, the unit risk factor may change as a result of new peer reviewed research.

We welcome any opportunity to provide additional information helpful to you or that would facilitate the process of identification.

We would appreciate our Findings and this transmittal letter being made a part of the final report.

Sincerely,

/s/

John R. Froines, Ph.D.  
Acting Chairman  
Scientific Review Panel

Enclosure

cc: Scientific Review Panel Members  
Michael Kenny, ARB  
Bill Lockett, ARB

Findings of the Scientific Review Panel on  
**THE REPORT ON DIESEL EXHAUST**  
as adopted at the Panel's April 22, 1998, Meeting

Pursuant to Health and Safety Code section 39661, the Scientific Review Panel (SRP/Panel) has reviewed the report *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant* by the staffs of the California Air Resources Board (ARB or Board) and the Office of Environmental Health Hazard Assessment (OEHHA) describing the public exposure to, and health effects of, diesel exhaust. The Panel members also reviewed the public comments received on this report.

Panel members participated in workshops devoted to discussion of the exposure and health issues associated with diesel exhaust in September 1994, January 1996, July 1997, and March 1998. The SRP reviewed the issues at its meetings in October 1997 and April 1998. A special meeting of the SRP was held on March 11, 1998, to hear testimony on health issues including the quantitative risk assessment from highly respected scientists invited by the Panel. Based on these reviews and information provided at scientific workshops and meetings, the SRP makes the following findings pursuant to Health and Safety Code section 39661:

**Exposure related conclusions**

1. Diesel exhaust is a complex mixture of gases and fine particles emitted by a diesel-fueled internal combustion engine.
2. The gaseous fraction is composed of typical combustion gases such as nitrogen, oxygen, carbon dioxide, and water vapor. However, as a result of incomplete combustion, the gaseous fraction also contains air pollutants such as carbon monoxide, sulfur oxides, nitrogen oxides, volatile organics, alkenes, aromatic hydrocarbons, and aldehydes, such as formaldehyde and 1,3-butadiene and low-molecular weight polycyclic aromatic hydrocarbons (PAH) and PAH-derivatives.
3. One of the main characteristics of diesel exhaust is the release of particles at a markedly greater rate than from gasoline-fueled vehicles, on an equivalent fuel energy basis. The particles are mainly aggregates of spherical carbon particles coated with inorganic and organic substances. The inorganic fraction primarily consists of small solid carbon (or elemental carbon) particles ranging from 0.01 to 0.08 microns in diameter. The organic fraction consists of soluble organic compounds such as aldehydes, alkanes and alkenes, and high-molecular weight PAH and PAH-derivatives, such as nitro-PAHs. Many of these PAHs and PAH-derivatives, especially nitro-PAHs, have been found to be potent mutagens and carcinogens. Nitro-PAH compounds can also be formed during transport through the atmosphere by reactions of adsorbed PAH with nitric acid and by gas-phase radical-initiated reactions in the presence of oxides of nitrogen.

4. Diesel exhaust includes over 40 substances that are listed by the United States Environmental Protection Agency (U.S. EPA) as hazardous air pollutants and by the ARB as toxic air contaminants. Fifteen of these substances are listed by the International Agency for Research on Cancer (IARC) as carcinogenic to humans, or as a probable or possible human carcinogen. Some of these substances are: acetaldehyde; antimony compounds; arsenic; benzene; beryllium compounds; bis(2-ethylhexyl)phthalate; dioxins and dibenzofurans; formaldehyde; inorganic lead; mercury compounds; nickel; POM (including PAHs); and styrene.
5. Almost all of the diesel particle mass is in the fine particle range of 10 microns or less in diameter (PM<sub>10</sub>). Approximately 94 percent of the mass of these particles are less than 2.5 microns in diameter. Because of their small size, these particles can be inhaled and a portion will eventually become trapped within the small airways and alveolar regions of the lung.
6. The estimated population-weighted average outdoor diesel exhaust PM<sub>10</sub> concentration in California for 1995 is 2.2 microgram per cubic meter ( $\mu\text{g}/\text{m}^3$ ). Several independent studies have reported similar outdoor air diesel exhaust PM<sub>10</sub> concentrations. The 1995 estimated average indoor exposure concentration is approximately 1.5  $\mu\text{g}/\text{m}^3$ .
7. The population time-weighted average total air exposure to diesel exhaust particle concentrations across all environments (including outdoors) is estimated to be 1.5  $\mu\text{g}/\text{m}^3$  in 1995. This total exposure estimate may underestimate many Californians' actual total exposure because it excludes elevated exposures near roadways, railroad tracks, and inside vehicles. Near-source exposures to diesel exhaust may be as much as five times higher than the 1995 population time-weighted average total air exposure. It also excludes other routes of exposure to diesel exhaust, such as ingestion and dermal absorption.
8. Diesel engine exhaust contains small carbonaceous particles and a large number of chemicals that are adsorbed onto these particles or present as vapors. These particles have been the subject of many studies because of their adverse effects on human health and the environment. A recent study conducted for the Health Effects Institute showed that, despite a substantial reduction in the weight of the total particulate matter, the total number of particles from a 1991-model engine was 15 to 35 times greater than the number of particles from a 1988 engine when both engines were operated without emission control devices. This suggests that more fine particles, a potential health concern, could be formed as a result of new technologies. Further study is needed since the extent of these findings only measured exhaust from two engines and engine technologies.
9. The major sources of diesel exhaust in ambient outdoor air are estimated to emit approximately 27,000 tons per year in 1995. On-road mobile sources (heavy-duty trucks, buses, light-duty cars and trucks) contribute the majority of total diesel exhaust PM<sub>10</sub> emissions in California. Other mobile sources (mobile equipment, ships, trains, and boats) and stationary sources contribute the remaining emissions.

10. Significant progress has been made as a result of federal and state regulations that have addressed particulate matter levels from diesel engines. Emissions of on-road mobile source diesel exhaust PM<sub>10</sub> in California are expected to decline by approximately 85 percent from 1990 to 2010 as a result of mobile source regulations already adopted by the ARB.
11. The results of a study funded by the ARB at the University of California, Riverside, indicate that the diesel exhaust from the new fuel tested contained the same toxic air contaminants as the old fuel, although their concentrations and other components may differ. Further research would be helpful to quantify the amounts of specific compounds emitted from a variety of engine technologies, operating cycles, and fuel to characterize better any differences between old and new fuels and technologies.

### **Health effects associated with diesel exhaust**

12. A number of adverse short-term health effects have been associated with exposures to diesel exhaust. Occupational exposures to diesel exhaust particles have been associated with significant cross-shift decreases in lung function. Increased cough, labored breathing, chest tightness, and wheezing have been associated with exposure to diesel exhaust in bus garage workers. A significant increase in airway resistance and increases in eye and nasal irritation were observed in human volunteers following one-hour chamber exposure to diesel exhaust. In acute or subchronic animal studies, exposure to diesel exhaust particles induced inflammatory airway changes, lung function changes, and increased the animals' susceptibility to infection.
13. A number of adverse long-term noncancer effects have been associated with exposure to diesel exhaust. Occupational studies have shown that there may be a greater incidence of cough, phlegm and chronic bronchitis among those exposed to diesel exhaust than among those not exposed. Reductions in pulmonary function have also been reported following occupational exposures in chronic studies. Reduced pulmonary function was noted in monkeys during long-term exposure. Histopathological changes in the lung of diesel-exposed test animals reflect inflammation of the lung tissue. These changes include dose-dependent proliferations of type II epithelial cells, marked infiltration of macrophages, plasma cells and fibroblasts into the alveolar septa, thickening of the alveolar walls, alveolar proteinosis, and focal fibrosis.
14. Studies have shown that diesel exhaust particles can induce immunological reactions and localized inflammatory responses in humans, as well as acting as an adjuvant for pollen allergy. Intranasal challenge with diesel exhaust particles in human volunteers resulted in increased nasal IgE antibody production and a significant increase in mRNA for pro-inflammatory cytokines. Co-exposure to diesel exhaust particles and ragweed pollen resulted in a nasal IgE response greater than that following pollen or diesel exhaust particles alone. Effects of intratracheal, intranasal, and inhalation exposures of laboratory animals are supportive of the findings in humans. These effects include eosinophilic

infiltration into bronchi and bronchioles, elevated IgE response, increased mucus secretion and respiratory resistance, and airway constriction.

15. Based on the animal studies, the U.S. EPA determined a chronic inhalation Reference Concentration value of  $5 \mu\text{g}/\text{m}^3$  for noncancer effects of diesel exhaust. This estimate takes into consideration persons who may be more sensitive than others to the effects of diesel exhaust. The report supports the recommendation of  $5 \mu\text{g}/\text{m}^3$  as the California Reference Exposure Level (REL) (Table 1). It should be noted that this REL may need to be lowered further as more data emerge on potential adverse noncancer effects from diesel exhaust.
16. Diesel exhaust contains genotoxic compounds in both the vapor phase and the particle phase. Diesel exhaust particles or extracts of diesel exhaust particles are mutagenic in bacteria and in mammalian cell systems, and can induce chromosomal aberrations, aneuploidy, and sister chromatid exchange in rodents and in human cells *in vitro*. Diesel exhaust particles induced unscheduled DNA synthesis *in vitro* in mammalian cells. DNA adducts have been isolated from calf thymus DNA *in vitro* following treatment with diesel exhaust particle extracts. DNA adducts have been shown to increase following inhalation exposure of rodents and monkeys to whole diesel exhaust. Elevated levels of DNA adducts have been associated with occupational exposure to diesel exhaust. Results of inhalation bioassays in the rat, and with lesser certainty in mice, have demonstrated the carcinogenicity of diesel exhaust in test animals, although the mechanisms by which diesel exhaust induces lung tumors in animals remains uncertain.
17. Over 30 human epidemiological studies have investigated the potential carcinogenicity of diesel exhaust. These studies, on average, found that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer. The lung cancer findings are consistent and the association is unlikely to be due to chance. These epidemiological studies strongly suggest a causal relationship between occupational diesel exhaust exposure and lung cancer.
18. Other agencies or scientific bodies have evaluated the health effects of diesel exhaust. The National Institute of Occupational Safety and Health first recommended in 1988 that whole diesel exhaust be regarded as a potential occupational carcinogen based upon animal and human evidence. The International Agency for Research on Cancer (IARC) concluded that diesel engine exhaust is probably carcinogenic to humans and classified diesel exhaust in Group 2A. Based upon the IARC findings, in 1990, the State of California under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) identified diesel exhaust as a chemical "known to the State to cause cancer." The U.S. EPA has proposed a conclusion similar to IARC in their draft documents. The 1998 draft U.S. EPA document concluded similarly that there was sufficient animal evidence of carcinogenicity and that the human evidence was limited.
19. There are data from human epidemiological studies of occupationally exposed populations which are useful for quantitative risk assessment. The estimated range of lung cancer risk (upper 95% confidence interval) based on human epidemiological data is  $1.3 \times 10^{-4}$  to 2.4

$\times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$  (Table 2). After considering the results of the meta-analysis of human studies, as well as the detailed analysis of railroad workers, the SRP concludes that  $3 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$  is a reasonable estimate of unit risk expressed in terms of diesel particulate. Thus this unit risk value was derived from two separate approaches which yield similar results. A comparison of estimates of risk can be found in Table 3.

20. Based on available scientific information, a level of diesel exhaust exposure below which no carcinogenic effects are anticipated has not been identified.
21. Based on available scientific evidence, as well as the results of the risk assessment, we conclude that diesel exhaust be identified as a Toxic Air Contaminant.
22. As with other substances evaluated by this Panel and after reviewing the field of published peer reviewed research studies on diesel exhaust, additional research is appropriate to clarify further the health effects of diesel exhaust. This research may have significance for estimating the unit risk value.
23. The Panel, after careful review of the February 1998 draft SRP version of the ARB report, *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant*, as well as the scientific procedures and methods used to support the data, the data itself, and the conclusions and assessments on which the Report is based, finds this report with the changes specified during our October 16, 1997, meeting and as a result of comments made at the March 11, 1998, meeting, is based upon sound scientific knowledge, methods, and practices and represents a complete and balanced assessment of our current scientific understanding.

For these reasons, we agree with the science presented in Part A by ARB and Part B by OEHHA in the report on diesel exhaust and the ARB staff recommendation to its Board that diesel exhaust be listed by the ARB as a Toxic Air Contaminant.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on April 22, 1998.

/s/

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John R. Froines, Ph.D  
Acting Chairman,  
Scientific Review Panel

**TABLE 1**  
**NONCANCER HEALTH VALUES APPROVED BY THE**  
**SCIENTIFIC REVIEW PANEL**  
**1998**

Compound	Health Value	Endpoint
Acetaldehyde	$9 \mu\text{g}/\text{m}^3$	Respiratory System
<b>Diesel Exhaust</b>	<b><math>5 \mu\text{g}/\text{m}^3</math></b>	<b>Respiratory System</b>
Inorganic Lead	$4.6 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$	Cardiovascular Mortality
Perchloroethylene	$35 \mu\text{g}/\text{m}^3$	Alimentary System (Liver)

$\mu\text{g}/\text{m}^3$ : microgram per cubic meter

**TABLE 2**

**CANCER POTENCIES APPROVED BY THE SCIENTIFIC REVIEW PANEL  
FROM 1984 TO 1998  
(in order of cancer potency)**

<b>Compound</b>	<b>Unit Risk (<math>\mu\text{g}/\text{m}^3</math>)<sup>-1</sup></b>	<b>Range (<math>\mu\text{g}/\text{m}^3</math>)<sup>-1</sup></b>
Dioxins	$3.8 \times 10^1$	$2.4 \times 10^1$ to $3.8 \times 10^1$
Chromium VI	$1.5 \times 10^{-1}$	$1.2 \times 10^{-2}$ to $1.5 \times 10^{-1}$
Cadmium	$4.2 \times 10^{-3}$	$2.0 \times 10^{-3}$ to $1.2 \times 10^{-2}$
Inorganic Arsenic	$3.3 \times 10^{-3}$	$6.3 \times 10^{-4}$ to $1.3 \times 10^{-2}$
Benzo[a]pyrene	$1.1 \times 10^{-3}$	$1.1 \times 10^{-3}$ to $3.3 \times 10^{-3}$
<b>Diesel Exhaust</b>	<b><math>3 \times 10^{-4}</math></b>	<b><math>1.3 \times 10^{-4}</math> to <math>2.4 \times 10^{-3}</math></b>
Nickel	$2.6 \times 10^{-4}$	$2.1 \times 10^{-4}$ to $3.7 \times 10^{-3}$
1,3-Butadiene	$1.7 \times 10^{-4}$	$4.4 \times 10^{-6}$ to $3.6 \times 10^{-4}$
Ethylene Oxide	$8.8 \times 10^{-5}$	$6.1 \times 10^{-5}$ to $8.8 \times 10^{-5}$
Vinyl Chloride	$7.8 \times 10^{-5}$	$9.8 \times 10^{-6}$ to $7.8 \times 10^{-5}$
Ethylene Dibromide	$7.1 \times 10^{-5}$	$1.3 \times 10^{-5}$ to $7.1 \times 10^{-5}$
Carbon Tetrachloride	$4.2 \times 10^{-5}$	$1.0 \times 10^{-5}$ to $4.2 \times 10^{-5}$
Benzene	$2.9 \times 10^{-5}$	$7.5 \times 10^{-6}$ to $5.3 \times 10^{-5}$
Ethylene Dichloride	$2.2 \times 10^{-5}$	$1.3 \times 10^{-5}$ to $2.2 \times 10^{-5}$
Inorganic Lead	$1.2 \times 10^{-5}$	$1.2 \times 10^{-5}$ to $6.5 \times 10^{-5}$
Perchloroethylene	$5.9 \times 10^{-6}$	$3.0 \times 10^{-7}$ to $1.1 \times 10^{-5}$
Formaldehyde	$6.0 \times 10^{-6}$	$2.5 \times 10^{-7}$ to $3.3 \times 10^{-5}$
Chloroform	$5.3 \times 10^{-6}$	$6.0 \times 10^{-7}$ to $2.0 \times 10^{-5}$
Acetaldehyde	$2.7 \times 10^{-6}$	$9.7 \times 10^{-7}$ to $2.7 \times 10^{-5}$
Trichloroethylene	$2.0 \times 10^{-6}$	$8.0 \times 10^{-7}$ to $1.0 \times 10^{-5}$
Methylene Chloride	$1.0 \times 10^{-6}$	$3.0 \times 10^{-7}$ to $3.0 \times 10^{-6}$
Asbestos	$1.9 \times 10^{-4}$ (per 100fiber/m <sup>3</sup> )	Lung: $11 - 110 \times 10^{-6}$ (per 100 fiber/m <sup>3</sup> ) Mesothelioma: $38 - 190 \times 10^{-6}$ (per 100 fiber/m <sup>3</sup> )

$\mu\text{g}/\text{m}^3$ : microgram per cubic meter

**TABLE 3**

**Comparison of Other Organizations' Estimated 95% Upper Confidence Limits of Lifetime Risk per  $\mu\text{g}/\text{m}^3$  Diesel Particulate Matter from Risk Assessments Based on Epidemiologic Data with OEHHA Estimates**

<b>Method</b>	<b>Unit Risk/Range</b>	<b>Basis of Assessment</b>	<b>Reference</b>
Epidemiologic analysis	$3 \times 10^{-4}$	based on smoking-adjusted pooled RR	Smith, 1998
<b>Epidemiologic analysis<sup>b</sup></b>	<b><math>3.6 \times 10^{-4}</math> to <math>2.4 \times 10^{-3}</math></b>	<b>case-control study of Garshick et al., 1987</b>	<b>OEHHA, Part B, Section 7.3.3</b>
<b>Epidemiologic analysis</b>	<b><math>2.8 \times 10^{-4}</math> to <math>1.8 \times 10^{-3}</math></b>	<b>cohort study of Garshick et al., 1988</b>	<b>OEHHA, Part B, Section 7.3.4</b>
<b>Epidemiologic analysis</b>	<b><math>1.3</math> to <math>7.2 \times 10^{-4}</math></b>	<b>cohort study, time varying conc., roof (3,50) pattern</b>	<b>OEHHA, Part B, Appendix D</b>
<b>Epidemiologic analysis</b>	<b><math>3.8 \times 10^{-4}</math> to <math>1.9 \times 10^{-3}</math></b>	<b>cohort study, time varying conc., ramp (1,50) pattern</b>	<b>OEHHA, Part B, Appendix D</b>
Epidemiologic analysis	$1.4 \times 10^{-3}$	London transport study <sup>c</sup>	Harris, 1983
Epidemiologic analysis	$2 \times 10^{-3}$	epidemiologic data of Garshick (top end of U.S. EPA's range)	U.S. EPA, 1998;
Epidemiologic analysis	$1.3 \times 10^{-4}$ to $1.3 \times 10^{-2}$	using smoking adjusted RR and exposures of 5 or $500 \mu\text{g}/\text{m}^3$	OEHHA, Part B, Section 7.3; bracketed risk bounds

a) Bolded values are included in OEHHA's range of risk.

b) Obtained by applying Harris' slope of  $5 \times 10^{-4} (\mu\text{g}/\text{m}^3 \times \text{yr})^{-1}$  to California life table.